REMARKS

Review and reconsideration of the Office Action of August 24, 2007 is respectfully requested in view of the above amendments and the following remarks.

The claims were amended to refer to a hard solid matrix. Support for the claim amendment can be found on paragraphs [00030] to [00035] of the specification as originally filed.

No new matter has been entered to the claims by the present amendment.

For the reasons set forth below, Applicants believe that the present set of claims is novel and not obvious over the cited art.

Office Action

Turning to the Office Action, the paragraphing of the Examiner is adopted.

(Detailed Action)

The Examiner indicated that Claims 7-8 stand withdrawn from further consideration as being drawn to a non-elected invention.

The position of the Examiner can be found on page 2 of the Office Action.

Applicants note that on a **previous** Office Action, the Examiner indicated that the method claims (Claims 7-8) will be **re-joint** to the product claims if the product claims are found

allowable and the method claims include all the limitations of the product claims.

Applicants respectfully request that the Examiner re-joint the method claims to the product claims if the product claims are found allowable.

Anticipation rejection

The Examiner rejects Claims 2-3, 6, 15, 16, and 19 under 35 U.S.C. 102(b) as being anticipated by McKibben US Patent No. 6,183,733.

The position of the Examiner can be found on pages 2-3 of the Office Action.

Applicants respectfully traverse.

For a reference to anticipate, it must contain all the elements of the Claim.

Applicants noted that the present set of claims includes two independent claims, namely, Claims 16 and 20. Claim 20 was not rejected in view of the McKibben reference.

The following remarks are addressed to the rejected independent Claim 16, because if this claim is not anticipated, it follows that none of the other rejected dependent claims are anticipated.

First, Applicants note that McKibben teaches a composition of attracting over-wintering boll weevils. In one embodiment, column 6, lines 50-58, the composition includes a polymer based insecticidal composition including a polymer, plant volatile, a

pheromone, and an insecticide. The reference also teaches the use of a plasticizer mixed by the polymer (Column 7, lines 13-16).

Regarding Claim 16

Compare with independent Claim 16 of the present set of claims, this reference fails to teach: 1) a composition containing a **vapor releasing** insecticide and 2) a **hard** solid matrix.

Point 1

Present Claim 16 is limited to the exclusive use of a vapor releasing insecticide, dichlorvos (DDVP). DDVP is very volatile and highly toxic by inhalation. See attachment C.

The present invention does not use just any organophosphate as an insecticide but uses **a vapor releasing insecticide** that has a vapor that is toxic to weevils.

The use of a vapor releasing insecticide is an important factor in the present invention because direct contact between the boll weevil and the invention is not required in order to kill the weevil and prevent its escape from the boll weevil trap.

McKibben provides a list of possible insecticides that can possibly be used. But the reference is silent regarding using **a vapor releasing** insecticide. Applicants note that the reference uses Malathion, guthion, and methyl parathion as insecticides. These insecticides are **not vapor releasing insecticides**.

The MSDS of Malathion indicated that the evaporation rate of **the chemical is negligible**. See attachment A.

The information found for methyl parathion indicated indicates that the chemical stays on the area where it is applied for several months. Thus, the chemical cannot be a vapor release insecticide. See attachment B.

Thus, Malathion, methyl parathion, and guthion are not vapor releasing insecticides.

In addition, it is clear from Column 7 lines 41 - 43, that McKibben only had in mind insecticides that **had to be ingested by the weevil** in order for killing to occur.

"Insecticides incorporated into the plastisol kill the boll weevils **that ingest it**. The pellets when applied in the field attract and induce insects to ingest particles of the pellets. Subsequently, the insects die or are rendered infertile, if insect growth regulators are used."

Thus, the reference fails to teach **vapor releasing insecticide**.

In the present invention the election of a vapor releasing insecticide is not a mere fact of choice. In the present invention, the Grandlure attracts the weevils and **the vapor release insecticide kills the weevils by direct contact with the device and through vapor action thus preventing their escape and aiding in accurately counting the captured insects**. Specifically, the dichlorovos speeds up the release of Grandlure

from the matrix, while the plasticizer slows the release of the Grandlure. The divergent forces of the dichlorovos and the plasticizer acting on the Grandlure creates a condition wherein the active ingredients are released simultaneously over a sustained period of time giving it a longer life than just using a pheromone and an insecticide in separate dispensers.

McKibben et al teaches a method for manufacturing a friable material designed to be eaten in order to kill a captured insect. **A friable substrate may not be desirable for a vapor releasing insecticide due to the following**: The rate of release of a vapor producing insecticide such as dichlorvos is dependent upon the surface area of the composition among other factors. A friable or crumbling material may have far more surface area than a solid and hard dispenser such as the one for the present invention. Thus, there is no reasonable expectation that a friable material would be suitable when used with a vapor releasing insecticide such as dichlorvos.

The only way a person skilled in the art could determine which insecticide to use in Applicant's composition is via experimentation.

Point 2

The McKibben references teaches on Column 7, lines 26-32, that his product is soft enough to have a **friable** surface

(easily crumbled) thus insects can chew and ingest the active ingredients.

The solid matrix of the present invention **is hard** and not friable.

A solid substrate is more efficient than a friable substrate because it can kill a captured insect merely due to the nearby presence of insect. Ingestion of the substrate is not required.

Regarding Claim 19

Furthermore, compared with Claim 19, the reference fails to teach curing the homogeneous mixture forming the hard solid matrix, which is made of all the active ingredients.

Applicants note that Claim 19 requires that the solid matrix containing the **homogeneous mixture** is cured between 100 to 300 **°F**.

The Examiner indicated that Column 7, lines 21-23, of the McKibben reference teaches curing the homogeneous mixture between 100 to 300 **°F**.

Applicants reviewed the reference and noted that the reference is curing the **polymer (PVC) to form the inner core to a temperature** between 170-180 **°C** (Column 7, lines 21-23). **After the dipping process is complete, a** solid matrix **is formed, then the product is cured in an oven at a temperature just high**

enough to achieve a solid, non-tacky surface with a friable surface) (Column 7, 24-30).

> "In the dip molding process, dip molds are heated to a temperature sufficient to provide full curing of the PVC to form an inner core. Typical temperatures are between **170 and 180° C.,** to provide a durable support structure and to provide for a controlled release of the pheromone and the plant volatiles. After dipping, the resulting tubular structure can be cured in an oven at a temperature just high enough to achieve a solid, non-tacky surface but low enough to provide a **friable surface** that insects can chew and ingest."

First, Applicants note that there are two different temperature scales: **°F (present invention) and °C (McKibben)**; thus, the temperature range in both references is different.

Second, the reference is curing the polymer (PVC) **alone** to a temperature between 170-180°C; the reference is silent regarding the temperature range for the solid matrix after the dipping process.

A person skilled in the art will understand that the characteristic of the final product will depend on the curing temperature and the ingredients of the mixture before the curing.

Accordingly, withdrawal of the rejection is respectfully requested.

Obviousness rejection

The Examiner rejects Claims 2-4, 6, 14-16, 19, and 20 under 35 U.S.C. 103(a) as being obvious over McKibben (US Patent No. 6,183,733) in view of Smith US Patent No. 5,888,930 and Rowe (US Patent No. 2,775,994).

The position of the Examiner can be found on pages 2-3 of the Office Action.

Applicants respectfully traverse for the same reasons set forth above and the following remarks.

Applicants noted that the present set of claims includes two independent claims, namely, Claims 16 and 20.

The following remarks are addressed to the rejected independent Claims 16 and 20, because if these claims are not obvious, it follows that none of the other rejected dependent claims are obvious.

The Examiner indicated that the rejection of record is reinstated with Smith showing equivalence of the instant dichlorvos with McKibben's use of malathion.

Applicant's position regarding the McKibben reference can be found above.

Applicants note that the Smith reference indeed shows a vapor release insecticide.

Combining the references (replacing the malathion insecticide of McKibben with the DDVP of Smith)

First, Applicants note that malathion (McKibben) is a **non-vapor release** insecticide. Second, DDVP (Smith) is **a vapor release** insecticide.

As indicated before, it is clear from Column 7 lines 41 - 43, that McKibben only had in mind insecticides that **had to be ingested by the weevil** in order for killing to occur.

Thus, there is not any technological motivation to replace **a non-vapor release** insecticide with **a vapor release** insecticide because the McKibben reference requires that the composition be **ingested** by the animal. If the insecticide used in McKibben is a vapor release insecticide, the efficacy of the insecticide will be loss prior to the contact with the animal.

McKibben et al teaches a method for manufacturing a friable material designed to be eaten in order to kill a captured insect. **A friable substrate may not be desirable for a vapor releasing insecticide due to the following**: The rate of release of a vapor producing insecticide such as dichlorvos is dependent upon the surface area of the composition among other factors. A friable or crumbling material may have far more surface area than a solid and hard dispenser such as the one for the present invention. Thus, there is no reasonable expectation that a friable material would be suitable when used with a vapor releasing insecticide such as dichlorvos.

The use of a vapor releasing insecticide is an important factor in the present invention because direct contact between the boll weevil and the invention is not required in order to kill the weevil and prevent its escape from the boll weevil trap.

Regarding Claim 20

This claim includes the close transitional phrase **consisting of;** this terminology is interpreted to mean that any embodiment that does not contain exactly (no more or no less than) the elements recited in the claims is not considered to be encompassed by the claim. The terminology may include the presence of trace amounts of additional components that are normally present as impurities.

The McKibben patent uses the **plant volatiles (kairomone)** eugenol (Claim 1) and myrcene (Claim 24). Plant volatiles **are one of McKibben's essential active ingredients,** as is discussed extensively in the McKibben patent in Column 3 lines 62- 72; and Column 4 lines 1-64. Also, each of McKibben's independent Claims 1, 10, 15, 18, 24 and 25 contain a plant volatile.
Thus, the composition of the reference contains no more of the elements than the elements recited in the claims.

Favorable consideration and early issuance of the Notice of

Allowance are respectfully requested. Should further issues remain prior to allowance, the Examiner is respectfully requested to contact the undersigned at the indicated telephone number.

Respectfully submitted,

Evelyn A. Defilló
Registration No. 45,630

Defillo & Associates, Inc.
P.O. Box 14104
Clearwater, FL 33766
727 772-5916

Date: **November 26, 2007**

CERTIFICATE OF MAILING

I hereby certify that the foregoing AMENDMENT F for U.S. Application No. 10/752,801 filed January 07, 2004, was deposited in first class U.S. mail, with sufficient postage, addressed to: Mail Stop Amendment, Commissioner for Patents, P. O. Box 1450, Alexandria, VA 22313-1450 on November 26, 2007.

Evelyn A. Defilló

Nu-Gro Corporation Inc.
10 Craig Street, Brantford, Ontario, N3R 7J1
TEL (519) 757-0077 FAX (519) 757-0080

MATERIAL SAFETY DATA SHEET

PRODUCT: Malathion UEV Insecticide

SECTION 1 - PRODUCT INFORMATION

P.C.P. Act Registration No.: 14597 **Code:** 11400

Chemical Name: Malathion: O,O-dimethyl-S-(1,2(di(ethoxycarbonyl)-ethyl) phosphorodithioate
Synonyms: Fyfanon
Chemical Family: Organophosphate
Product Use: Insecticide
TDG Classification: Environmentally Hazardous Substance, Liquid, n.o.s. (Malathion Solution), Class 9.2, UN3082, PG III

SECTION 2 - HAZARDOUS INGREDIENTS

HAZARDOUS INGREDIENTS	WEIGHT %	CAS REG #	LD50 (mg/Kg)	T.W.A. - T.L.V.
Malathion	95.0	121-75-5	Oral, rat 1375 Dermal,rat 4400	LC50 1.6 mg/L
Calculated LD50 for mixture			1448	

SECTION 3 - PHYSICAL DATA

Physical State: Liquid
Specific Gravity: 1.23
Boiling Point: 156 - 157 °C
Vapour Pressure: 3.4×10^{-6} mm Hg @ 25 °C
% Volatiles: Negligible
Solubility in Water: 148.2 mg/L
Appearance\Odour: Pale yellow liquid with slightly aromatic odour
pH: No Data
Freezing/Melting Point: Approx. 3 °C
Vapour Density: 11.4 (air=1)
Evaporation Rate: Negligible

SECTION 4 - FIRE AND EXPLOSION

Flash Point: 163 °C Pensky Martens
Autoignition Temperature: No Data
Lower Explosion Limit %: No Data
Upper Explosion Limit %: No Data
Fire Extinguishing Media: Foam, Carbon Dioxide, Dry Chemical, Water Fog
Fire Fighting Procedures: Wear self-contained Breathing Apparatus and impervious clothing. Minimize the amount of water used and contain the run-off.
Other Fire or Explosion Hazards: Malathion should never be heated above 55 °C.

SECTION 5 - REACTIVITY DATA

Stability: Stable
Hazardous Polymerization: Will Not Occur
Conditions to Avoid: Elevated temperatures, will decompose at 100 °C and above.
Incompatibility (Materials to Avoid): Strong alkalies and oxidizers.
Hazardous Decomposition Products: Thermal decomposition may produce dimethylsulfide, Sulphur oxides, carbon oxides, phosphoropentoxide, hydrogen sulfide and nitrogen oxides.

SECTION 6 - HEALTH HAZARD DATA

Acute Effects of Overexposure: May cause headache, nausea, vomiting, abdominal cramps, weakness, blurred vision, pin-point pupils, tightness in chest, laboured breathing, sweating, muscle spasms and coma.
Effects of Chronic Exposure: Prolonged exposure may cause dermatitis.
Other Health Effects: Malathion is a cholinesterase inhibitor. NOTE TO PHYSICIAN: The antidote is atropine. Large doses at 2 to 4 mg intravenously or intramuscularly as soon cyanosis is overcome. Repeat at 5 to 10 minute intervals until signs of atropinization appear.

SECTION 7 - FIRST AID PROCEDURES

Inhalation: Move victim to fresh air and restore breathing if required. OBTAIN MEDICAL ADVICE.
Skin Contact: Flush skin with running water and thoroughly wash with soap and water. If irritation persists seek medical attention.
Ingestion: Give 1 to 2 glasses water (200 to 500 ml) to dilute material. Do Not Induce Vomiting. OBTAIN MEDICAL ADVICE. . If spontaneous vomiting occurs, lean victim forward with head down to avoid breathing of vomitus, rinse mouth and administer more water. Never give anything by mouth to an unconscious person.
Eye Contact: Flush eyes with running water for 20 minutes. Hold eyelids open during flushing. If irritation persists seek Medical Attention.

SECTION 8 - PREVENTIVE MEASURES

Respiratory Protection: NIOSH/MSHA approved respirator.
Eye Protection: Chemical safety glasses.
Skin Protection: Chemical resistant gloves.
Other Personal Protective Equipment: Coveralls.
Engineering Controls: Local exhaust or ventilation.
Handling Procedures and Equipment: Avoid breathing vapours, contact with eyes, skin and clothing. Wash thoroughly after use.
Storage Requirements: Store in cool, dry, ventilated area. Keep out of reach of children and pets.
Storage Temperature: Min: 5 °C Max: 40 °C

SECTION 9 - ENVIRONMENTAL PROTECTION DATA

Spill and Leak Procedures: Stop leak, contain spill by diking and absorb with suitable absorbent and transfer into waste container for disposal. Clean area with detergent and water, absorb wash and place in waste container. Remove any contaminated soil for proper disposal.
Waste Disposal: Dispose of empty container in household garbage. Dispose of waste product in accordance with Local, Provincial or Federal government regulations.
Environmental Effects: Do not contaminate local water supplies or environments.

The information contained herein is considered accurate and offered only as a guide to the handling of this specific material. This information, does not relate to its use with any other material or product or in any process. No warranty, expressed or implied is made regarding the accuracy of the data or the performance of the material by Nu-Gro Corporation Inc. This Material Safety Data Sheet is valid for three Years from Issue Date.

Prepared by: Technical Department **Issue Date:** March 31, 2000

PUBLIC HEALTH STATEMENT
METHYL PARATHION
CAS#: 298-00-0

Division of Toxicology **September 2001**

This Public Health Statement is the summary chapter from the Toxicological Profile for Methyl Parathion. It is one in a series of Public Health Statements about hazardous substances and their health effects. A shorter version, the ToxFAQs™, is also available. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present. For more information, call the ATSDR Information Center at 1-888-422-8737.

This public health statement tells you about methyl parathion and the effects of exposure. The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Methyl parathion has been found in at least 16 of the 1,585 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which methyl parathion is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance, or by skin contact. If you are exposed to methyl parathion, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS METHYL PARATHION?

Methyl parathion is a pesticide that is used to kill insects on crops. Usually, it is sprayed on the crops. Methyl parathion comes in two forms: a pure form of white crystals and a technical-grade solution (brownish liquid), which contains methyl parathion (80%) and inactive ingredients in a solvent. The technical-grade methyl parathion smells like rotten eggs or garlic. Methyl parathion is a manufactured chemical, so it is found in the environment only as a result of its manufacture or use. Methyl parathion has been manufactured in the United States since 1952 and has been used to kill insects on many types of crops since this time. Because methyl parathion can be dangerous to humans, the EPA has restricted how it can be used and applied. Methyl parathion must be sprayed on crops from the air or from the ground in certain ways to minimize the danger of being exposed, and only trained people are allowed to spray methyl parathion. Methyl parathion is no longer used on food crops commonly consumed by children, and the maximum amount of methyl parathion that can be present as a residue on specific crops is regulated (see Section 1.9). In these ways, exposure to methyl parathion can be controlled and accidental exposures can be prevented.

attachment B

PUBLIC HEALTH STATEMENT
METHYL PARATHION
CAS#: 298-00-0

1.2 WHAT HAPPENS TO METHYL PARATHION WHEN IT ENTERS THE ENVIRONMENT?

Once methyl parathion is introduced into the environment from spraying on crops, droplets of methyl parathion in the air fall on soil, plants, or water. While most of the methyl parathion will stay in the areas where it is applied, some can move to areas away from where it was applied by rain, fog, and wind. Methyl parathion stays in the environment from a few days to several months. It is degraded to other chemical compounds by water, sunlight, and bacteria found in soil and water. On soil, methyl parathion sticks to the soil, and then is rapidly degraded by bacteria. It generally does not leach through the ground and end up in the groundwater. In water, methyl parathion breaks down quickly by the action of the water, bacteria in the water, and sunlight. In water and air, methyl parathion is broken down by sunlight to form a more toxic product called methyl paraoxon. If concentrated amounts of methyl parathion are present in soil, such as at landfills and hazardous waste sites, methyl parathion does not degrade as fast.

1.3 HOW MIGHT I BE EXPOSED TO METHYL PARATHION?

Most people are not exposed to methyl parathion in the air they breathe or on things they touch, unless they live next to areas being sprayed. The people who are at the greatest risk of being exposed to methyl parathion are those who work with this chemical. These include farm workers, chemical sprayers, and people who work in factories that make methyl parathion. They are exposed to methyl parathion on things they touch where it can pass through their skin, or by breathing it after it has been sprayed. Overexposure to methyl parathion may cause severe poisoning or death. Persons may be exposed to dangerous amounts if they go into fields too soon after spraying. The people most likely to be exposed to methyl parathion can be protected by wearing special clothing and breathing equipment and by staying out of sprayed fields for at least 2 days.

Individuals can also be exposed if they live near landfills where methyl parathion has been dumped or near water containing methyl parathion that washes off nearby land or that is accidentally spilled. The greatest amounts of methyl parathion are expected to be present near or on the farms where methyl parathion is used. After spraying, some methyl parathion can be transported by the wind or fog to areas away from where it is used, but the amounts present at these locations are not expected to be at dangerous levels. In 1988, one location in Mississippi had groundwater that contained 88 parts of methyl parathion per billion parts of water (ppb). More recent studies of water samples taken near where methyl parathion was sprayed indicate methyl parathion is not found in the groundwater. The risk of exposure to methyl parathion from drinking groundwater appears to be low, but the EPA is currently examining this issue.

Methyl parathion is approved only for use on crops. The maximum amount of methyl parathion residue allowed by the Food and Drug Administration (FDA) and EPA on crops used as food is 0.1–1 ppm. The FDA has monitored the food supply for

pesticides for a number of years. FDA purchases many kinds of foods through Market Basket Surveys and analyzes them for residue levels of pesticides. These FDA studies allow scientists to estimate the daily intake of pesticides. Generally, the FDA monitoring studies conclude that the U.S. food supply contains only very small amounts of pesticides that are not a concern. However, there have been some reports of the illegal use of methyl parathion inside homes.

1.4 HOW CAN METHYL PARATHION ENTER AND LEAVE MY BODY?

Methyl parathion can enter your body if you eat food or drink water containing it; if you swim, bathe, or shower in contaminated water; if you touch recently sprayed plants or soil; if you touch contaminated soil near hazardous waste sites; or if you breathe air that contains methyl parathion, such as near factories or recently sprayed farm fields (or in recent accounts of the illegal use of methyl parathion, if you breathe air or touch contaminated surfaces inside homes where methyl parathion has been used to kill insects). By any means of exposure, methyl parathion goes into your body quickly and gets into your blood. From your bloodstream, methyl parathion goes to your liver, brain, and other organs. Your liver changes some of methyl parathion to a more harmful chemical called methyl paraoxon. Both methyl parathion and methyl paraoxon can bind to enzymes of your nerves within minutes or hours. Your liver breaks down methyl parathion and methyl paraoxon into less harmful substances. These less harmful substances leave your body in urine within hours or days.

1.5 HOW CAN METHYL PARATHION AFFECT MY HEALTH?

Methyl parathion interferes with the normal way that the nerves and brain function. Exposure to very high levels of methyl parathion for a short period in air or water may cause death, loss of consciousness, dizziness, confusion, headaches, difficult breathing, chest tightness, wheezing, vomiting, diarrhea, cramps, tremors, blurred vision, and sweating. Some people who have been exposed to substances similar to methyl parathion have experienced changes in mental state that lasted several months after exposure to high levels of these substances ended. If people are exposed to levels of methyl parathion below those that affect nerve function, few or no health problems seem to occur. There is no evidence that methyl parathion causes birth defects in humans or affects the ability of humans to produce children. There is also no proof that methyl parathion causes cancer in people who are regularly exposed, such as farmers and pesticide applicators.

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests. One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body; for some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat

research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

Animal studies show effects of methyl parathion similar to those seen in people. In addition, short-term high exposure of animals to methyl parathion caused decreased heart rate. This may be the result of methyl parathion's effects on the nerves that control the heart. Methyl parathion decreased the ability of animals to fight infections in some studies, but not in others. It is not known whether any of these effects occur in people. It is not known whether methyl parathion affects the ability of animals to reproduce. Studies in animals have not shown that methyl parathion causes cancer.

1.6 HOW CAN METHYL PARATHION AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans. Children are likely to be exposed to methyl parathion in the same ways as adults, mainly by eating foods or drinking milk or water that contain residues of this chemical. Because of their smaller weight, children's intake of methyl parathion per kilogram of body weight may be greater than that of adults. The FDA and EPA permit residues of pesticides to be present in crops used as food, and these amounts are considered to be safe. The EPA, however, has recently used stricter regulations and has canceled the use of methyl parathion on food crops commonly eaten by children. As a result, the exposure of children to methyl parathion from foods will be very small.

Children are affected by methyl parathion in the same manner as adults. Exposure to high levels of methyl parathion, even for short periods, may result in changes in the nervous system, leading to headaches, dizziness, confusion, blurred vision, difficulty breathing, vomiting, diarrhea, loss of consciousness, and death (see also Section 1.5 for a more complete description of how methyl parathion affects human health). It is not known whether children are more sensitive to the effects of methyl parathion than adults. There is some indication that young rats may be more sensitive than adults to nervous system effects.

There is no evidence in humans that methyl parathion causes birth defects. Birth defects have not been seen when methyl parathion was given to animals by mouth, but minor birth defects did occur in one study in which high doses were injected into pregnant animals. It is not known whether these effects occur in people. It is unlikely that people would be exposed by breathing, touching, or eating as much methyl parathion as was injected in the animal studies. Animal studies have also shown that methyl parathion can be transferred from a pregnant mother to the developing fetus. Methyl parathion caused changes in the behavior of young animals whose mothers were given methyl parathion during pregnancy, and this effect needs to be studied more. Methyl parathion has been detected in small amounts in breast milk, but only in a few localities in central Asia. Studies of mother animals fed methyl parathion show that methyl parathion can be transferred into their milk and their nursing newborn babies.

PUBLIC HEALTH STATEMENT
METHYL PARATHION
CAS#: 298-00-0

1.7 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO METHYL PARATHION?

If your doctor finds that you have been exposed to significant amounts of methyl parathion, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

The only approved use of methyl parathion is on crops, including crops used as foods. Effective December 31, 1999, the EPA cancelled the use of methyl parathion on many kinds of crops used as foods because of a concern for exposure risks to children and to workers. This action will reduce the risks to families of methyl parathion exposure from food. The general population is not likely to be exposed to large amounts of methyl parathion. The populations living in the areas where methyl parathion is used on crops, however, may be exposed to greater amounts of methyl parathion. Methyl parathion is often detected in foods and air samples collected where methyl parathion is used. People who live close to areas of methyl parathion use also may be exposed to larger amounts of methyl parathion, because small amounts of the pesticide will move from the place where it is used to nearby areas. These exposures may include such things as touching contaminated plants, breathing the mist formed from the sprayed chemical, drinking contaminated water, or eating recently sprayed fruits and vegetables. People who are most likely to receive the highest exposures are those who work in the factories that make methyl parathion, workers who spray it on crops, and farmers. Entry of methyl parathion into the body after contact with the skin is expected to be the major exposure pathway for those working in these operations. Breathing the mist containing methyl parathion may also occur.

Families can reduce the risk of exposure to methyl parathion in the soil, on plants, or in the air by staying away from fields that have been recently sprayed. If families wait at least 4–5 days before entering sprayed fields, then the amount of methyl parathion present in the air or on plants is expected to be small. Families should also be aware that sometimes methyl parathion has been illegally sprayed inside the home to kill insects. Your children may be exposed to methyl parathion if an unqualified person applies pesticides containing it around your home. In some cases, the improper use of pesticides banned for use in homes has turned homes into hazardous waste sites. Make sure that any person you hire is licensed and, if appropriate, certified to apply pesticides. Your state licenses each person who is qualified to apply pesticides according to EPA standards and further certifies each person who is qualified to apply "restricted use" pesticides. Ask to see the license and certification. Also ask for the brand name of the pesticide, a Material Safety Data Sheet (MSDS), the name of the product's active ingredient, and the EPA registration number. Ask whether EPA has designated the pesticide "for restricted use" and what the approved uses are. This information is important if you or your family react to the product. If you buy over-the-counter pesticide products to apply yourself, be sure the products are in unopened pesticide containers that are labeled and contain an EPA registration number. Carefully follow the instructions on the label. If you plan to spray inside,

make sure the pesticide in intended for indoor use. If you feel sick after a pesticide has been used in your home, consult your doctor or local poison control center.

1.8 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO METHYL PARATHION?

Several medical tests can determine whether you have been exposed to methyl parathion. The first medical test measures methyl parathion in your blood or measures 4-nitrophenol, which is a breakdown product of methyl parathion, in your urine. These tests are only reliable for about 24 hours after you are exposed because methyl parathion breaks down quickly and leaves your body. These tests cannot tell whether you will have harmful health effects or what those effects may be. The next medical test measures the levels of a substance called cholinesterase in your blood. If cholinesterase levels are less than half of what they should be and you have been exposed to methyl parathion, then you may get symptoms of poisoning. However, lower cholinesterase levels may also only indicate exposure and not necessarily harmful effects. The action of methyl parathion may cause lower cholinesterase levels in your red blood cells or your blood plasma. Such lowering, however, can also be caused by factors other than methyl parathion. For example, cholinesterase values may already be low in some people, because of heredity or disease. However, a lowering of cholinesterase levels can often show whether methyl parathion or similar compounds have acted on your nerves. Cholinesterase levels in red blood cells can stay low for more than a month after you have been exposed to methyl parathion or similar chemicals.

1.9 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH). Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors. Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for methyl parathion include the following:
NIOSH recommends that a person not be exposed in the workplace to more than 0.2 mg/m3 of methyl

parathion for a 10-hour workday, 40-hour workweek.

According to EPA, the following levels of methyl parathion in drinking water are not expected to cause effects that are harmful to health: 0.3 mg/L for 1 or 10 days of exposure for children, 0.03 mg/L for longer term exposure for children, and 0.002 mg/L for lifetime exposure of adults.

It is illegal to use methyl parathion indoors. Methyl parathion is approved only for use on agricultural crops. In 1999, EPA canceled the use of methyl parathion on many food crops, particularly those consumed by children, such as apples, peaches, pears, carrots, and peas, and also canceled nonfood uses such as ornamental plants and nursery stock uses. Methyl parathion use is still allowed on other crops eaten by people or by farm animals. A maximum of 0.1–1 ppm of methyl parathion is allowed in or on the other crops (fruits, vegetables, nuts, and grains) that may be eaten by people.

1.10 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333

Information line and technical assistance:

Phone: 888-422-8737
FAX: (770)-488-4178

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

To order toxicological profiles, contact:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 800-553-6847 or 703-605-6000

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2001. Toxicological profile for methyl parathion. Update. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

E X T O X N E T

Extension Toxicology Network

Attachment C

A Pesticide Information Project of Cooperative Extension Offices of Cornell University, Michigan State University, Oregon State University, and University of California at Davis. Major support and funding was provided by the USDA/Extension Service/National Agricultural Pesticide Impact Assessment Program.

Pesticide
Information
Profile

Dichlorvos

Publication Date: 9/93

TRADE OR OTHER NAMES

Apavap, Benfos, Cekusan, Cypona, Derriban, Derribante Devikol, Didivane, Duo-Kill, Duravos, Elastrel, Fly-Die, Fly-Fighter, Herkol, Marvex, No-Pest, Prentox, Vaponite, Vapona, Verdican, Verdipor, Verdisol. Trade names used outside of the U.S. include Doom, Nogos, and Nuvan (2).

REGULATORY STATUS

A Special Review of dichlorvos was initiated in February 1988 because EPA determined that the registered uses of dichlorvos may pose a risk of cancer as well as inadequate margins of safety for cholinesterase inhibition and liver effects to exposed persons (12). The Special Review was not complete as of March 1992 (10). Products containing dichlorvos must bear the signal words "Danger-Poison" (2).

INTRODUCTION

Dichlorvos is used to control household, public health, and stored product insects. It is effective against mushroom flies, aphids, spider mites, caterpillars, thrips, and white flies in greenhouse, outdoor fruit, and vegetable crops (2). Therapeutically, dichlorvos is used to treat a variety of parasitic worm infections in dogs, livestock and humans. Dichlorvos can be fed to livestock to control botfly larvae in the manure. It acts against insects as both a contact and a stomach poison (2). Dichlorvos is available in aerosol and soluble concentrate formulations (2). It is used as a fumigant (2) and has been used to make pet collars and pest strips (3).

Dichlorvos is one of a class of insecticides referred to as organophosphates. These chemicals act by interfering with the activities of cholinesterase, an enzyme that is essential for the proper working of the nervous systems of both humans and insects. Please refer to the Toxicology Information Brief on cholinesterase-inhibition for a more detailed description of this topic.

In 1955, it was discovered that crystalline trichlorfon, another organophosphate pesticide, gave off a vapor which was capable of killing insects. That vapor was dichlorvos, which has since been developed for insect control in enclosed spaces (3).

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Dichlorvos is highly toxic by inhalation, dermal absorption and ingestion (9). Because dichlorvos is volatile, inhalation is the most common route of exposure. As with all organophosphates, dichlorvos is readily absorbed through the skin. Skin which has come in contact with this material should be washed immediately with soap and water and all contaminated clothing should be removed.

Acute illness from dichlorvos is limited to the effects of cholinesterase inhibition. Compared to poisoning by other organophosphates, dichlorvos causes a more rapid onset of symptoms, which is often followed by a similarly rapid recovery (3). This occurs because dichlorvos is rapidly metabolized and eliminated from the body. Persons with reduced pulmonary (lung) function, convulsive disorders, liver disorders, or recent exposure to cholinesterase inhibitors will be at increased risk from exposure to dichlorvos. Alcoholic beverages may enhance the toxic effects of dichlorvos. High environmental temperatures or exposure of dichlorvos to visible or UV light may enhance its toxicity (9).

Dichlorvos is mildly irritating to skin (9). Concentrates of dichlorvos may cause burning sensations, or actual burns (6). Dichlorvos can be very toxic if it is not immediately washed off, but instead left on the skin long enough for it to become absorbed through the skin and into the bloodstream. One man nearly died after spilling 4 ounces of a 3% oil solution of dichlorvos on his lap. He did not wash it off. Another man only became nauseous and dizzy after spilling a similar amount on his arm. He washed off the dichlorvos with soap and water (6). Do not use organic solvents to remove dichlorvos from the skin (DLA/DOD Hazardous Mat'ls Info. System #0014-29- 438-0000. 1982).

The organophosphate insecticides are cholinesterase inhibitors. They are highly toxic by all routes of exposure. When inhaled, the first effects are usually respiratory and may include bloody or runny nose, coughing, chest discomfort, difficult or short breath, and wheezing due to constriction or excess fluid in the bronchial tubes. Skin contact with organophosphates may cause localized sweating and involuntary muscle contractions. Eye contact will cause pain, bleeding, tears, pupil constriction, and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours. These may include pallor, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, eye pain, blurred vision, constriction or dilation of the eye pupils, tears, salivation, sweating, and confusion. Severe poisoning will affect the central nervous system, producing incoordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids, and eventually paralysis of the body extremities and the respiratory muscles. In severe cases there may also be involuntary defecation or urination, psychosis, irregular heart beats, unconsciousness, convulsions and coma. Death may be caused by respiratory failure or cardiac arrest (9).

Some organophosphates may cause delayed symptoms beginning 1 to 4 weeks after an acute exposure which may or may not have produced immediate symptoms. In such cases, numbness, tingling, weakness and cramping may appear in the lower limbs and progress to incoordination and paralysis. Improvement may occur over months or years, but some residual impairment will remain (9).

The administration of slow-release formulations of dichlorvos to domestic animals to treat for internal parasites has caused some inhibition of cholinesterase and mild symptoms such as nausea or diarrhea, but no serious signs of illness. Repeated, small doses generally have no effect on treated animals. Doses of up to 4 mg/kg of a slow release formulation, given to cows to reduce flies in their feces, had no

visibly adverse effects on the cows. Blood tests of these cows indicated cholinesterase inhibition (3).

Dichlorvos is very volatile, meaning that it readily forms vapors which may be inhaled. Inhalation is the most common way to be exposed to dichlorvos. Low, repeated doses may be non-toxic. High doses of dichlorvos may be very toxic, especially if inhalation exposure is continuous (6). Dichlorvos produces irritating gases, such as phosphorous and chlorine oxides, when heated (NIH/EPA 1984).

Eye protection should be worn when handling dichlorvos. Application of 1.67 mg/kg in rabbits' eyes produced mild redness and swelling, but no injury to the cornea (9). Dichlorvos may cause eye burns. Organophosphates cause the pupils to constrict (pin point pupils).

The amount of a chemical that is lethal to one-half (50%) of experimental animals fed the material is referred to as its acute oral lethal dose fifty, or LD50. The oral LD50 for dichlorvos in mice is 61 to 175 mg/kg, 100 to 1090 mg/kg in dogs, 15 mg/kg in chickens, 25 to 80 mg/kg in rats, 157 mg/kg in pigs, and 11 to 12.5 mg/kg in rabbits (2, 6, 9). The dermal LD50 for dichlorvos in rats is 70.4 to 250 mg/kg, 206 mg/kg in mice, and 107 mg/kg in rabbits (2, 3, 6, 9).

The lethal concentration fifty, or LC50, is that concentration of a chemical in air or water that kills half of the experimental animals exposed to it for a set time period. The 4-hour LC50 for dichlorvos in rats is 15 mg/m3, and 13 mg/m3 in mice (9).

CHRONIC TOXICITY

Feeding studies indicate that a dosage of dichlorvos very much larger than doses which inhibit cholinesterase are needed to produce illness. Rats tolerated dietary doses as high as 62.5 mg/kg/day for 90 days with no visible signs of illness, while a dietary level of 0.25 mg/kg/day for only 4 days produced a reduction in cholinesterase levels (3).

Rats were exposed to air concentrations of 0, 0.05, 0.5 and 5 mg/m3 of dichlorvos over a 5 week period. Rats in the 0.5 and 5 mg/kg groups exhibited significantly decreased cholinesterase activity in the plasma, red blood cells, and brain. The NOEL for this study was 0.05 mg/m3. In dogs fed dietary doses of 0.0095, 0.016, 0.16, 1.6 or 12.5 mg/kg/day for 2 years, decreased red blood cell cholinesterase activity, increased liver weights and increased liver cell size occurred in the two highest doses tested. The NOEL was 0.08 mg/kg/day (12). Chronic exposure to dichlorvos will cause fluid to build up in the lungs (pulmonary edema) (NIH/EPA; OHM/TADS 1984).

Repeated or prolonged exposure to organophosphates may result in the same effects as acute exposure including the delayed symptoms. Other effects reported in workers repeatedly exposed include impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise has also been reported (9).

Reproductive Effects

When male and female rats were given a diet containing 100 ppm (5 mg/kg/day) dichlorvos just before mating, and with this dosage continued through pregnancy and lactation for females, there were no effects on reproduction or on the survival or growth of the offspring, even though severe cholinesterase inhibition occurred in the mothers and significant inhibition occurred in the offspring. The same results were observed in a 3-generation study with rats fed dietary levels up to (25 mg/kg/day) (3). Once in the bloodstream, dichlorvos may cross the placenta (9).

Teratogenic Effects

A dose of 12 mg/kg was not teratogenic in rabbits and did not interfere with reproduction in any way. There was no evidence of teratogenicity when rats and rabbits were exposed to air concentrations of up to 6.25 mg/m3 throughout pregnancy. Dichlorvos was not teratogenic when given orally to rats (3).

Mutagenic Effects

Dichlorvos can bind to molecules such as DNA. For this reason, there has been extensive testing of dichlorvos for mutagenicity. Several studies reviewed by EPA have shown dichlorvos to be a mutagen (12). Dichlorvos is reported positive in the Ames mutagenicity assay (Mut. Res. 87:211 (1981); 76:169 (1980); 40 (1):19 (1976) and in other tests involving bacterial or animal cell cultures. However no evidence of mutagenicity has been found in tests performed on live animals. Its lack of mutagenicity in live animals may be due to rapid metabolism and excretion of dichlorvos (3).

Carcinogenic Effects

Dichlorvos has been classified as a possible human carcinogen by EPA because of the results of tests on rats and mice (11). When dichlorvos was administered by gavage to mice for 5 days per week for 103 weeks at doses of 10 or 20 mg/kg to males and 20 or 40 mg/kg to females, there was an increased incidence of benign tumors in the lining of the stomach at the high dose for both sexes. When rats given daily doses of 0, 4 or 8 mg/kg for five days per week for 103 weeks, there was an increased incidence of benign tumors of the pancreas and of leukemia in male rats at both doses. At the highest dose, there was also an increased incidence of benign lung tumors in males. In female rats, there was an increase in the incidence of benign tumors of the mammary gland (12). No tumors caused by dichlorvos were found in rats fed up to 25 mg/kg/day for 2 years or in dogs fed up to 11 mg/kg/day for 2 years. No evidence of carcinogenicity was found when rats were exposed to air containing up to 5 mg/m3 for 23 hours/day for 2 years (3). A few tumors were found in the esophagus of mice given dichlorvos orally, even though tumors of this kind are normally rare (9).

Organ Toxicity

Dichlorvos primarily affects the nervous system through cholinesterase inhibition, by which there is a deactivation of cholinesterase, an enzyme required for proper nerve functioning.

Dichlorvos causes fluid to accumulate in the lungs (6). Liver enlargement has occurred in pigs maintained for long periods of time on high doses (500 ppm) (3, 6). Dichlorvos caused adverse liver effects in dogs (12). Lung hemorrhages may occur (14). Cholinesterase inhibition may affect the nervous system. In mice, a single oral dose of 40 micrograms (ug)/kg caused changes in the testes. In male rats, repeated doses caused abnormalities in the tissues of the lungs, heart, thyroid, liver and kidneys (9).

Fate in Humans and Animals

Amongst the organophosphates, dichlorvos is remarkable for its rapid metabolism and excretion by mammals. Dichlorvos was not detected in the blood of rats, mice or people after exposure to atmospheric concentrations of up to 17 times that normally reached for insect control in homes. Exposure of rats to 11 mg/m3 (250 times the normal exposure) for 4 hours was required before dichlorvos was detectable in the rats. Even then, it was detected only in the kidneys. At 90 mg/m3 (2000 times normal exposure), dichlorvos was detected in most tissues of the rat. Following exposure to 50

mg/m3, the half-life for dichlorvos in the rat kidney was 13.5 minutes. The reason for this rapid disappearance of dichlorvos is the presence of degrading enzymes in both tissues and blood plasma. From the gastrointestinal tract, dichlorvos is absorbed into the portal blood, rather than into the general bloodstream. From the portal blood, it is moved to the liver where it is rapidly detoxified. Thus poisoning by nonlethal doses of dichlorvos is usually followed by rapid detoxification in the liver and recovery. Rats given oral or dermal doses at the LD50 level either died within one hour of dosing or recovered completely (3, 6).

Dichlorvos does not accumulate in body tissues and has not been detected in the milk of cows or rats, even when the animals were given doses high enough to produce symptoms of severe poisoning (3).

ECOLOGICAL EFFECTS

Effects on Birds

Dichlorvos is highly toxic to birds including ducks and pheasants (4, 8). The LD50 for wild birds fed dichlorvos is 12 mg/kg (NIOSH RTECS Online File #82/8110).

Effects on Aquatic Organisms

UV light makes dichlorvos more toxic to aquatic life by 5-150 times (15). NIH/EPA found the grass shrimp to be more sensitive to dichlorvos than the sand shrimp, hermit crab and mummichog (in that order) (1984). For ocean-dwelling species they found: scud > Atlantic silverside > striped killfish > striped mullet > bluehead > American eel > northern puffer; where ">" indicates a greater sensitivity to dichlorvos. The 96-hour LC50 for dichlorvos in fathead minnow is 11.6 mg/l, 0.9 mg/l in bluegill, 5.3 mg/l in mosquito fish, 0.004 ppm in sand shrimp, 3.7 ppm in mummichogs, and 1.8 ppm/96 hours in American eels (NIH/EPA 1984). The 24-hour LC50 for dichlorvos in bluegill sunfish is 1.0 mg/l (2).

Dichlorvos does not significantly bioaccumulate in fish (4).

Effects on Other Animals (Nontarget species)

Dichlorvos is toxic to bees (2).

ENVIRONMENTAL FATE

Breakdown of Chemical in Soil and Groundwater

Dichlorvos does not adsorb to soil particles and it is likely to contaminate groundwater. When spilled on soil, dichlorvos leached into the ground with 18 to 20% penetrating to a depth of 30 cm within 5 days. In soil, dichlorvos is subject to hydrolysis and biodegradation. Volatilization from moist soils is expected to be slow. Half-lives of 7 days were measured on clay, sandy-clay, and loose sandy soil (4).

Dichlorvos is rapidly broken down in the air and in damp media such as soil. The pH of the media determines the rate of breakdown. Alkaline soils, water, etc., show rapid breakdown, whereas acidic media shows slow degradation. For instance, at a pH of 9.1 the half-life of dichlorvos is about 4.5 hours. At a pH of 1 (very acidic), the half- life is 50 hours (8). Dichlorvos is non-persistent.

Breakdown of Chemical in Water

In water dichlorvos remains in solution and does not adsorb to sediments. It degrades primarily by hydrolysis, with a half-life of approximately 4 days in lakes and rivers. This half-life will vary from 20 to 80 hours between pH 4 and pH 9. Hydrolysis is slow at pH 4 and rapid at pH 9 (4, 5). Biodegradation may occur, especially under acidic conditions which slow hydrolysis, or where populations of acclimated micro-organisms exist, as in polluted waters. Volatilization from water is expected to be slow. The volatilization half-life from river and pond waters have been estimated at 57 and over 400 days respectively (4).

Breakdown of Chemical in Vegetation

Except for cucumbers, roses, and some chrysanthemums, plants tolerate dichlorvos very well (5).

PHYSICAL PROPERTIES AND GUIDELINES

Dichlorvos is a colorless to amber liquid with a mild chemical odor. Dilute dichlorvos breaks down rapidly in the presence of moisture. Concentrated forms are readily decomposed by strong acids and bases (3). Dichlorvos is stable under normal temperatures and pressures, but it may pose a moderate fire hazard if exposed to heat or flame. It may hydrolyze on contact with moisture, and may decompose in the presence of strong acids or bases (3, 9). Thermal decomposition of dichlorvos will release toxic oxides of phosphorus and carbon, toxic and corrosive chlorides and toxic phosgene gas. Dichlorvos is corrosive to iron and steel. It may attack materials such as plastics, rubber and coatings (9). Other metals (stainless steel, aluminum, nickel) are resistant if no water is present.

Dichlorvos increases the effects of malathion (5). Alcoholic beverages promote the absorption of dichlorvos into the bloodstream (8).

Persons who work with organophosphate materials for long periods of time should have frequent blood tests of their cholinesterase levels. If the cholinesterase level falls below a critical point, no further exposure should be allowed until it returns to normal (13).

Protective clothing must be worn when handling dichlorvos. Before removing gloves, wash them with soap and water. Always wash hands, face and arms with soap and water before smoking, eating or drinking.

After work, remove all work clothes and shoes. Shower with soap and water. Wear only clean clothes when leaving the job. Wash contaminated clothing and equipment with soap and water after each use. Keep contaminated work clothes separate from regular laundry.

Exposure Guidelines:

1 mg/m3 OSHA TWA (skin) (9)
0.1 ppm (0.9 mg/m3) ACGIH TWA (skin) (9)
1 mg/m3 NIOSH Recommended TWA (skin) (9)
Air concentrations of 200 mg/m3 are immediately dangerous to life or health (9).
PADI: 8 x 10 to the minus 4 power mg/kg/day, based on a 2-year dog feeding study (12)

Physical Properties

CAS #:	62-73-7
Specific gravity:	1.44 (60 degrees /60 degrees F) (2)
Solubility in water:	1 g/100g at 25 degrees C (17)
Solubility:	Miscible in non-polar solvents such as dichloromethane, 2-propanol and toluene (2, 17). Soluble in ethanol, chloroform, acetone, and kerosene (1, 5). Miscible in alcohol and in aromatic and chlorinated hydrocarbon solvents. Solubility in kerosene and mineral oils is about 3% (3).
Boiling point:	140 degrees C at 20 mm Hg (17); 117 degrees C at 11 mm Hg (2); 35 degrees C at 0.05 mm Hg (3); 183 degrees F (84 degrees C) (9)
Flash point:	>175 degrees F (>80 degrees C) (2, 16), practically non-flammable (17).
Vapor pressure:	0.01 mm Hg at 30 degrees C (18)
Chemical class/use:	Organophosphate insecticide

BASIC MANUFACTURER

Amvac Chemical Corp.
4100 E. Washington Blvd.
Los Angeles CA 90023

Review by Basic Manufacturer

Comments solicited: January, 1992.
Comments received: April, 1992.

REFERENCES

1. Farm Chemicals Handbook. Ed by R.T. Meister. Meister Publishing Co., Willoughby, OH. 1987.
2. Meister, R.T. (ed.). 1992. Farm Chemicals Handbook '92. Meister Publishing Company, Willoughby, OH.
3. Hayes, W.J. and E.R. Laws (ed.). 1990. Handbook of Pesticide Toxicology, Vol. 3, Classes of Pesticides. Academic Press, Inc., NY.
4. Howard, P.H. (ed.). 1989. Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Vol. III: Pesticides. Lewis Publishers, Chelsea, MI.
5. The Pesticide Manual. 8th Ed. Ed. by C.R. Worthing. British Crop Protection Council. 1987.
6. Pesticides Studied in Man. Wayland Hayes, Jr. Williams and Wilkins, Baltimore, MD. 1982.
7. Herbicides. 2nd Ed. Vols. 1, 2. Kearney. 1975.
8. The Agrochemicals Handbook. The Royal Society of Chem. 1983.
9. Occupational Health Services, Inc. 1991 (May 15). MSDS for Dichlorvos. OHS Inc., Secaucus, NJ.
10. US Environmental Protection Agency. 1992 (March). Status of Pesticides in Reregistration and Special Review (H-7508W). Prevention, Pesticides and Toxic Substances, US EPA, Washington,

DC.

11. _____. 1991 (Feb. 13). Dichlorvos; Revocation of tolerance and food additive regulation. Federal Register 56 (30): 5788-9.
12. _____. 1988 (Feb. 24). Dichlorvos; Initiation of Special Review. Federal Register 53 (36): 5542-9.
13. Cheminova Agro A/S. 1991 (June 11). Material Safety Data Sheet : Dimethoate. Cheminova, Lemvig, Denmark.
14. Pesticides and Human Health. Hallenbeck & Cunningham-Burns. Springer-Verlag.
15. TOXNET. 1985. National library of medicine's toxicology data network. Hazardous Substances Databank. Public health Service. National Institute of Health. U.S. Department of Health and Human Services. Bethesda, MD: NLM.
16. National Fire Protection Association (NFPA). Fire Protection Guide. Hazardous Materials. 1978.
17. Merck Index 9th Ed. 1976.
18. Handbook of Analytical Toxicology. Sunshine. 1969.

To Top

For more information relative to pesticides and their use, please contact the PMEP staff at:

Pesticide Safety Education Pgm.
5123 Comstock Hall
Cornell University
Ithaca, NY 14853-0901
(607) 255-1866

Pesticide Sales & Use Reporting Database Grp.
Cornell University
20 Thornwood Dr. #106
Ithaca, NY 14850
(607) 257-5706

Cornell University

This site is supported, in part, by funding from the Northeastern IPM Center

Questions regarding the development of this web site should be directed to the PMEP Webmaster